

Supplementary Table 1: Selected Genes Evaluated for Methylation Analysis with Locus, Published Frequency of Methylation and Biological Significance in AML.

Gene (Number of AML Samples Tested)	Percent Methylation (27)	Locus	Significance
<i>CDH1</i> (254)	40-60	16q22.1	Independently associated with adverse outcome in AML. (5, 28)
<i>CEBPα</i> (70)	0-10	19q13.1	Myeloid transcription factor, mutations associated with M1,2 AMLs. Deficient mice show blocks in granulocyte differentiations. Deletions associated with good prognosis AML. (29)
<i>CEBPδ</i> (80)	20-40	8p11.2- p11.1	Gene discovery studies in AML patient samples and in 5-azacytidine treated U937 cell lines have shown frequent methylation of this gene. (30)
<i>CTNNA1</i>	15	5q31	Suppression provides a growth advantage in MDS/AML. (31)
<i>ER</i> (289)	40-60	6q25.1	Hypermethylation of ER in MDS is associated with poor prognosis. (5)
<i>FHIT</i> (220)	40-60	3p14.2	TSG, associated with disease progression in MDS and AML. Associated with high white blood cell counts at diagnosis. (32, 33)
<i>MGMT</i> (40)	0-20	10q26	Methylation rare in AML but may predict a subset of patients with sensitivity to temozolamide. More frequent in samples from patients who have relapsed AML.(34, 35)
<i>MLH1</i> (20)	20-40	3p21.3	Methylation rare in AML patient samples.(34)
<i>p15</i> (697)	60-80	9p21	Methylated in MDS (poor risk), associated with 7q deletions. Potentially associated with increased risk of relapse in AML. (4, 5, 36, 37)
<i>p73</i> (28)	7	1p36	Sequence homology to p53, important for mismatch repair and apoptosis pathways. (16)
<i>SOCS1</i> (231)	40-60	16p13.2	Downregulates JAK/STAT signaling, associated with t(15;17), and other karyotypic abnormalities. Associated with high risk MDS phenotypes, and increased leukemic transformation. (38, 39)

Supplementary Table 2: Primers for Methylated and Unmethylated Reactions

Genes	Unmethylated Forward	Unmethylated Reverse	Bp Size
<i>CDHI</i>	TGG TTG TAGTTATGTATTTATTTTATAGTGGTGT	ACA CCA AATACAATCAAATCAAACCAA A	120
<i>CEBP-α</i>	TTG TTGGGTATAAAAATTGGGTTGGT	AAA ATTCTCCCA ACATAACAAACCTCA	149
<i>CEBP-δ</i>	GGG GTGTTTTTGTGGTGTG	GGG GTGTTTTTGTGGTGTG	120
<i>CTNNA1</i>	AACACCCATCTACTTCAA ACCTCTAAA ATTTAACA	TTTAAG GATTTTTTTGGTTTGGTTTGAGAGATTGTG	115
<i>DAPK</i>	GGA GGATAGTTGGATTGAGTTAATGTT	CAA ATCCCTCCCAAAACACCAA	106
<i>ER</i>	TGTGTTTATGAGTTTAATGTTGTGGTT	AAA AAAACCCCCCAA ACCATT	124
<i>FHIT</i>	GGGTGTGGGTTTGGGTTTTATGT	CCATAA ACAACA CCA ACC CCA CTA AA	72
<i>MLH1</i>	TTTTGATGTAGATGTTTTATTAGGGTTGT	ACCACCTCATCATAACTACCCACA	124
<i>MGMT</i>	TTTGTGTTTTGATGTTTGTAGGTTTTTGT	AACTCCACACTCTTCCAAAAACAAAACA	93
<i>p15^{INK4B}</i>	GGTTGGTTTTTATTTTGTAGAGTGAGGT	AACCACTAACCACAAA ATACAAACACA	80
<i>p73</i>	AGG GGATGTAGTGA AATTGGGGTTT	ATCACA ACCCCA AACATCAACATCCA	69
<i>SOCS1</i>	TTGAGTTGTTGGAGTATTATGTGGTGGT	CACTAACAACACAACCTCCTACAACAACCA	100
Genes	Methylated Forward	Methylated Reverse	Bp Size
<i>CDHI</i>	TGTAGTTACGTATTTATTTTATAGTGGCGTC	CGAATACGATCGAATCGAACCG	112
<i>CEBP-α</i>	GTCGGGTATAAAAAGTTGGGTCGGC	ATTCTCCCGACATAACGAACCTCG	144
<i>CEBP-δ</i>	GCGTTTTCGCGGTGTC	AACCGAACTCTACGTCCAAACG	110
<i>CTNNA1</i>	GCCCGTCTACTT CGAACCTCTAAAATTTAACG	ATTTTTTCGGTTTGGTTTCGAGAGATCGC	106
<i>DAPK</i>	GGATAGTCGGATCGAGTTAACGTC	CCCTCCCAAACGCCGA	98
<i>ER</i>	ACGAGTTTAAACGTCGCGGTC	ACCCCCCAAACCGTTAAAAC	110
<i>FHIT</i>	GCGGGTTTGGGTTTTTACGC	CGACGCCGACCCCACTAAA	61
<i>MLH1</i>	ACGTAGACGTTTTTATTAGGGTCGC	CCTCATCGTAACTACCCGCG	115
<i>MGMT</i>	TTTCGACGTTTCGTAGGTTTTTCGC	GCACTCTCCGAAAACGAAACG	81
<i>p15^{INK4B}</i>	GGTTTTTATTTTGTAGAGCGAGGC	TAACCGCAAAATACGAACGCG	68
<i>p73</i>	GGACGTAGCGAAATCGGGGTTT	ACCCCGAACATCGACGTCCG	60
<i>SOCS1</i>	TGTTGGAGTATTACGTGGCGGC	CGACACAACCTCCTACAACGACCG	88